

Methods: An intravascular line (jugular vein) was placed in 4 anesthetized mice. Blood pressure and heart rate were monitored. Microbubbles (DMP-115, Aerosomes[®]) were diluted in 1/10 TTE was performed using a 13 MHz probe. A midventricular parasternal short axis view was obtained. Injections of contrast (0.05–0.1 ml of the solution) were performed and imaging acquired in the fundamental mode, linear post-processing. Images were digitized, consecutive end-systolic frames selected and videointensity (VI) measured in myocardial walls on each frame. The time of appearance of the contrast in myocardial walls (T1) was quantified in relation to seconds after appearance in left ventricle (LV).

Results: There was no significant effect of the contrast injections on mean blood pressure or heart rate (141 ± 15 versus 141 ± 13 mmHg, 503 ± 93 versus 510 ± 91 bpm). Contrast appearance was well visualized on all 15 injections in the left ventricle and in the myocardial walls (Table). Quantification of VI in the inferior wall was variable due to attenuation.

	Baseline (VI)	Peak contrast (VI)	T1 (sec)
LV cavity	20 ± 10	74 ± 12 [*]	0
Anterior wall	29 ± 2	56 ± 7 [*]	1.4 ± 0.7
Lateral wall	26 ± 7	47 ± 10 [*]	0.5 ± 0.3
Inferior wall	37 ± 6	51 ± 10	NA
Septal wall	18 ± 6	40 ± 7 [*]	0.6 ± 0.5

^{*} p < 0.003 versus baseline

Conclusion: Contrast echocardiography with DMP-115 is feasible and safe in mice. Contrast injections do not change hemodynamics and offer the potential for quantitation of myocardial perfusion in small animal models.

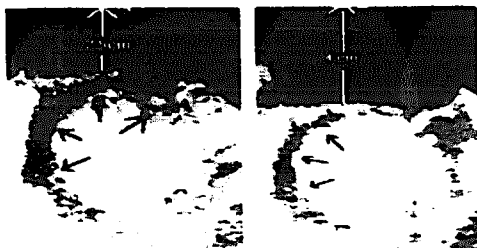
1179-138 Effect of Ultrasound Transducer Depth Setting in Identifying the Presence and Spatial Extent of Myocardial Perfusion Defects During Rest and Dobutamine Stress With Intermittent Harmonic Imaging

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Background: Intermittent harmonic ultrasound imaging (IHI) during a continuous intravenous infusion (CIVI) of microbubbles (MB) may potentially quantify myocardial perfusion abnormalities (MPA). Measurements of the spatial extent (SE) of these MPA depends on homogenous destruction of MB within the region of interest which may not occur. We hypothesized that uneven MB destruction due to beam attenuation prior to reaching the myocardium could alter quantitative measurements of a MPA with IHI.

Methods: We measured the SE of perfusion defects observed during a CIVI of perfluorocarbon exposed MB following left anterior descending (LAD) occlusion or during dobutamine induced ischemia (DII) in the setting of a significant LAD stenosis (50% diameter) in five open chest dogs. Measurements were made when the perfusion bed was at either a 2–3 or 4–5 cm standoff (SO) from the transducer. The SE of the MPA was correlated with post mortem risk area (RA).

Results: Risk area during LAD occlusion with IHI at a SO of 2–3 cm was 7.7 ± 1.3 cm², and was significantly closer to true RA (8.4 ± 1.5 cm²) than when the transducer was at a SO of 4–5 cm (4.2 ± 0.9 cm², p < 0.005, ANOVA). MPA during DII were significantly smaller when SO was increased to 4–5 cm (p < 0.05 compared to 2–3 cm SO; Figure).



Conclusions: We conclude that ultrasound beam attenuation can reduce the size of MPA observed with IHI during a CIVI of MB and, therefore, may reduce the sensitivity of this technique in detecting perfusion abnormalities in larger patients or in the inferior and posterior myocardium.

1179-139 Endocardial/epicardial Blood Flow Ratio Can Be Measured Using Venous Infusion of Microbubbles

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Background: Endocardial/epicardial (EER) can be measured with bolus injections of microbubbles in situations where endocardial myocardial blood volume (V) is decreased, such as with endocardial infarction. There is controversy, however, whether it can be measured in other settings. Unlike a bolus injection, where only V can be measured, venous infusions allow measurement of both V and microbubble velocity (v). We used this approach to measure EER in a model of coronary stenosis not associated with infarction.

Methods: Varying degrees of LAD stenosis were created in 12 open-chest dogs. Microbubbles were administered as a continuous infusion at a defined rate and concentration, and videointensity versus pulsing interval curves were obtained using intermittent harmonic imaging. From these curves, V and v were derived for individual pixels in each image. The product Vv denotes myocardial blood flow (MBF) in that pixel, which was then averaged over the endocardial and epicardial regions to derive EER. EER was also independently measured with radiolabeled microspheres. Data were acquired prior to and after induction of hyperemia.

Results: There was an excellent linear relation ($r = 0.81$, $p < 0.001$, $n = 24$) between EER using Vv versus microsphere data. The relation with V alone was poor ($r = 0.20$, $p = 0.35$), which indicates a dissociation between MBF and V distal to a stenosis.

Conclusion: The transmural distribution of MBF can be assessed using venous infusion of microbubbles, but only when both myocardial blood volume and microbubble velocity are measured.

1179-140 Reperfusion Injury Can Be Detected by Microbubble Persistence During Myocardial Contrast Echocardiography

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Background: We have previously shown that albumin microbubbles persist within the myocardium in the presence of microvascular endothelial injury. We, therefore, hypothesized that this phenomenon could be used to detect the presence of reperfusion injury.

Methods: Flow to the LAD was interrupted in 15 dogs for periods varying from 15 to 60 min, followed by 60 min of reperfusion. In 12 of these dogs, myocardial contrast echocardiography (MCE) was performed using constant rate and dose injections of Albunex[®] into the LAD prior to flow interruption and at 5, 15, 30, and 60 min following reflow. The fraction of persisting microbubbles was estimated from the tail of the time-intensity curves. In the remaining dogs, electron microscopy was performed with cationized ferritin labeling to look at the glycocalyx prior to flow cessation (1 dog) and following 15 min (1 dog) and 45 min (1 dog) of reperfusion.

Results: There was no microbubble adherence prior to flow interruption. It was observed in all but 1 dog after reflow. On an average, 5 ± 3% of the injected microbubbles were seen to persist within the myocardium after reflow. The magnitude of persistence correlated significantly ($p < 0.001$) with the duration of ischemia, but not that of reflow. Electron microscopy revealed no glycocalyx damage prior to flow interruption, but significant glycocalyx disruption after reflow.

Conclusions: Microvascular endothelial injury during reperfusion can be detected using MCE. Microbubble persistence is associated with glycocalyx damage in this setting. It may be possible in the future to examine the effect of interventions aimed at reducing reperfusion injury using this technique. These findings may potentially expand the role of MCE in acute myocardial infarction.

1179-141 Regional Myocardial Perfusion in Experimental Subarachnoid Hemorrhage

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Background: Subarachnoid Hemorrhage (SAH) is vanably associated with ECG changes, LV dysfunction, and contraction band necrosis (CBN) of the myocardium. Whether myocardial hypoperfusion causes these lesions remains unknown. In 5 open chest dogs, we studied the regional myocardial circulation in SAH.

Methods: Baseline evaluation included hemodynamic measurements (CO, LA pressure, BP), bloodwork (blood gas, CPK MB, catecholamines), ECG, 2D echo, and coronary angiography. Regional blood flow was assessed by 1) myocardial contrast echocardiography (MCE - triggered, harmonic mode) using aortic root injections of Albunex[®] and 2) injection of radiolabeled microspheres into the left atrium. SAH was created by injecting 0.4 cc/kg of